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MINDFULNESS, AORTIC WAVE REFLECTION, AND ARTERIAL STIFFNESS

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MINDFULNESS, AORTIC WAVE REFLECTION, AND ARTERIAL STIFFNESS

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Table of Contents

List of Figures

List of Tables

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List of Abbreviations

AIx	aortic augmentation index
AIx@75	aortic augmentation index normalized to 75 heart beats per minute
AP	augmentation pressure
aPL	aortic pulsatile load
BMI	body mass index
cfPWV	carotid-femoral pulse wave velocity
CVD	cardiovascular disease
DAP	diastolic arterial pressure
ECG	electrocardiogram
GAD	generalized anxiety disorder
HIC	high-income countries
HPA	hypothalamic-pituitary-adrenal
HR	heart rate
LMIC	low- and middle-income countries
MAP	mean arterial pressure.
MBSR	mindfulness-based stress reduction
PP	pulse pressure
PWA	pulse wave analysis
PWV	pulse wave velocity
SAP	systolic arterial pressure
STAI	state trait anxiety inventory

Abstract

Cardiovascular disease and hypertension are leading causes of death worldwide. The mitigation of high blood pressure is essential in decreasing the prevalence of cardiovascular-related deaths worldwide. Stress and anxiety are known to play a role in augmenting blood pressure in individuals of all ages. This increase in pressure can result in premature stiffening of large arteries in systemic circulation. Mindfulness is an ancient, non-secular practice which aids in stress reduction. Decentering, an aspect of mindfulness, involves accepting thoughts as transient rather than permanent associations. The purpose of this project was to examine the relationship between cardiovascular health and mindfulness practices. In **Study 1**, we investigated how a one-hour session of mindfulness meditation affected arterial stiffness and other cardiovascular variables. In **Study 2**, we compared inexperienced meditators' inherent ability to decenter with their arterial stiffness. We hypothesized that an acute meditation session would improve cardiovascular variables in Study 1. In Study 2, we postulated that individuals who are better able to decenter would have a slower pulse wave velocity and more elastic arteries. Our results from **Study 1** suggest that an acute session of meditation can significantly decrease aortic pulsatile load. In **Study 2**, we concluded that a greater ability to decenter is correlated with slower pulse wave velocity. These new findings support many previous studies that suggest that mindfulness practices are a beneficial lifestyle modification that can positively impact cardiovascular health.

1. Literature Review

The literature review of this thesis aims to first provide a brief synopsis of cardiovascular health and hypertension, the effect of stress and anxiety on the cardiovascular system, and the associated physiological mechanisms of the stress response. A short summary of the current knowledge of aortic stiffness, the systemic effects of arterial stiffness, wave reflection, and the associated measurement variables will be discussed. Then, a general overview of mindfulness meditation, including the efficacy of acute practices and decentering, will be presented. Finally, the literature review will include a general summary and proposed hypothesis for the two research studies.

1.1 Cardiovascular Health and Hypertension

Cardiovascular and other circulatory diseases are currently the most prevalent causes of death for people worldwide. Cardiovascular disease (CVD) plagues populations in all countries regardless of income. Between 1990 and 2013, individuals living in low- and middle-income countries (LMIC) experienced an increase in the proportion of deaths attributable to CVD at a younger age, while high-income countries (HIC) saw a slight decrease, likely due to improvements in primary and secondary prevention and care (62). Despite the trend, more individuals still die from CVD in HICs compared to LMICs.

Hypertension is the number one attributable risk for death worldwide (13). In 2017, the American Heart Association edited their guidelines for diagnosing hypertension. Elevated blood pressure is systolic blood pressure between 120-129 mmHg

and diastolic blood pressure less than 80 mmHg. Stage 1 hypertension includes systolic blood pressure between 130-139 mmHg and diastolic blood pressure between 80-89 mmHg, while stage 2 hypertension is systolic pressure greater than 140 mmHg or diastolic greater than 90 mmHg.

Suboptimal blood pressure has been associated with coronary artery disease, stroke, renal disease, and heart failure (15), making hypertension a multifaceted systemic pathology. Additionally, hypertension may also cause damage to the delicate microvasculature of target organs, which typically function at a lower resistance (68). Hypertension is often deemed the "silent killer," as up to 30% of adults are unaware of having high blood pressure (13). This presents a variety of problems and an increased risk for individuals who may not regularly check their blood pressure. Some epidemiological predictors and risk factors for developing hypertension include obesity, high sodium intake, increased arterial stiffness, and uncontrolled stress (41, 68).

In an attempt to mitigate the prevalence of hypertension and CVD-related deaths, the World Health Organization has set sustainable development goals with the intention of diminishing CVD-related deaths by 1/3 by the year 2030 (54). Other than pharmacological intervention and common lifestyle modifications such as exercise or diet, the maintenance of stress levels may be an appropriate adjunct treatment option for individuals with hypertension, which will be discussed in further detail later.

1.1.1 Cardiovascular System and HPA Axis

The stress response is an essential process in the human body. Evolutionarily, the cascade of events occurring during a stressful situation is physiologically necessary in

order to survive physical and dangerous threats (65). The hypothalamic-pituitary-adrenal (HPA) axis is an essential stress pathway which ultimately ends with the production and release of the glucocorticoid cortisol. Cortisol is an essential stress hormone that increases immediate energy availability in the body.

The mechanism of action for the HPA axis involves three primary structures: the hypothalamus, the pituitary gland, and the adrenal gland. In response to stressful stimuli, the hypothalamus releases corticotropin releasing hormone, which stimulates the anterior pituitary gland to secrete adrenocorticotropic hormone. This hormone stimulates the adrenal glands to release cortisol as a part of the stress response.

While the release of cortisol is essential in an acute stress response, issues arise when there is prolonged release of cortisol due to chronic stress, including suppression of the immune system, impairment of glucagon and insulin secretion, increases in pro-inflammatory cytokines, and increases in oxidative stress (23). An increase in inflammatory markers is an independent risk factor of premature death from cardiovascular disease (39). These cytokines, in conjunction with increased blood pressure from stress, can cause a multitude of negative effects in the vasculature, including endothelial damage, an atherosclerotic lipid profile, and smooth muscle cell hyperplasia (8). This, in turn, promotes atherosclerosis and increases in arterial stiffness.

1.1.2 Cardiovascular Health, Stress, and Anxiety

Acute and chronic psychosocial stressors are often associated with increased risk of cardiovascular events and hypertension (41, 59, 67). These stressors can include stress from work, life events (such as the death of a family member), anxiety, and emotional

regulation. Additionally, coping strategies may influence the impact of these life stressors (20). From a meta-analysis, individuals who reported elevated perceived stress had a 27% higher risk of CHD than those who had lower perceived stress (59). Stress from work, social isolation, and emotional stress is associated with an increased relative risk of CHD, as well as the risk for triggering acute cardiac events such as myocardial infarction (67). Life stress is also correlated with increases in arterial stiffness, partly due to the development of anxiety or depressive disorders (9).

Anxiety, specifically trait anxiety, has been strongly and positively correlated with hypertension (30, 40, 41). In fact, this association may be bidirectional - those with hypertension are more likely to have anxiety, while individuals with anxiety are more likely to be hypertensive (41). Anxiety is thought to affect cardiovascular health primarily through the HPA axis and increased sympathetic response, which leads to a cascade of physiological events including the increased release of stress hormones (catecholamines), endothelial damage, hypertension, atherosclerosis, and CHD (14). In hypertensive individuals, trait anxiety was positively correlated with elevated blood pressure (40) as well as emotional reactivity (30). These associations provide insight on the potential impact anxiety may have on the cardiovascular system.

In addition to stress and anxiety, unregulated emotional response may affect cardiovascular health. Previous survivors of acute myocardial infarction who scored higher in state and trait anger (anger as an emotional response and as a personality trait, respectively) were statistically more at risk of myocardial ischemia from emotional stress (56). In healthy females, higher anger scores were predictive of increased mean blood

pressure and ambulatory arterial stiffness (44), which indicates that effectively targeting the mitigation of anger may be a mode in which to reduce negative cardiovascular effects.

Not only is the reduction of stress imperative, but the way in which it is done is also an important consideration. Task-oriented coping involves purposeful thought or planning to solve a problem, while emotional-orientated coping may include rumination and emotions oriented toward the individual rather than the problem. Individuals who utilize a task-oriented coping mechanism, considered to be a functional coping style, have a significantly lower risk of hypertension compared to those who use an emotional-oriented style (20). Thus, developing positive, task-oriented coping methods in individuals may be a protective measure against hypertension. An example of a possible task-oriented coping strategy is mindfulness meditation, as individuals are tasked with the constant focus of the present moment. This link between psychological state and coping mechanism with CVD is an important focus for the continued effort to reduce the mortality of heart-related diseases.

1.2 Aortic Stiffness

The arteries are both capacitance and resistance vessels in the body. They receive blood from the heart to deliver to tissues while also functioning as pressure regulators. Arteries have a thick tunica media layer which contains smooth muscle. In response to sympathetic stimulation these muscles contract, decreasing the diameter of the vessel lumen and therefore increasing hemodynamic pressure.

Large arteries, and the smaller more distal arterioles, have structural differences that relate to their specific functions. In particular, the large arteries (such as the proximal

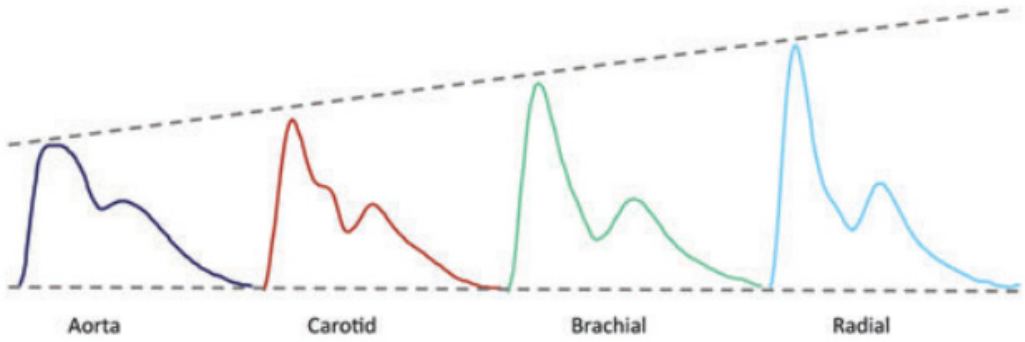

Figure 1. Waveform differences throughout vasculature. As the pulse wave moves through smaller vessel lumens, there is an increase in the systolic pressure represented by the larger initial inflection point in the waveform.

aorta) are composed of greater amounts of elastin while more distal vessels contain more of the stiffer collagen fibers (10). This leads to a phenomenon known as systolic pressure amplification. As the pulse wave moves through progressively smaller arteries and arterioles, systolic pressure increases which is demonstrated through increased systolic peaks on pressure waveforms (46).

The elastin in the large arteries permits the vessels to expand in order to accommodate the blood received from the left ventricle during systole, then slowly recoils to propagate the steady flow of blood. This aids in mitigating excess pulsatility that would arise from the intermittent pumping of the left ventricle. This is a protective feature that supports the more delicate microvasculature of organs with high blood flow (i.e., kidney), as these structures typically function with lower arterial resistance (12).

Arteries tend to progressively stiffen over time, a process that is considered a result of normal aging. This stiffening occurs linearly with age beginning in the 20-29

year age group and continues to increase throughout life (1). The changes in arterial stiffness longitudinally are also associated with increases in systolic blood pressure, mean arterial pressure, and pulse pressure, with a decrease in diastolic blood pressure (15). However, the premature stiffening of large arteries is especially problematic, as the stiffening of the large arteries can lead to the development of systolic hypertension, increases in carotid intima-media thickness, decreases in coronary perfusion with increases in afterload, increases in the pulsatility experienced by the microvasculature, and other detrimental hemodynamic dysfunction (12, 68). The relationship with hypertension and arterial stiffness may also be bidirectional, as hypertension has been observed to accelerate aortic degeneration leading to similar pressure changes from normal aging (52). These developments can eventually lead to different pathologies in various systems. In the heart, arterial stiffness is associated with left ventricle hypertrophy, which is present in heart failure (72). Along with age, arterial stiffness can be influenced by smoking, high BMI, and inactivity (12, 43).

Objectively quantifying the stiffness of large arteries is an early detectable way to assess structural and functional changes within vessel walls (10). Two of the most common and reliable methods to noninvasively measure the arterial pulse wave and arterial stiffness are through pulse wave analysis (PWA) and pulse wave velocity (PWV) via applanation tonometry. This will be discussed in further detail in the next headings.

1.2.1 Aortic Wave Reflection (Pulse Wave Analysis)

Forward traveling pulse waves from the left ventricle toward systemic circulation also transmit a reflected wave as pulses arrive at bifurcations or through changes in vessel

diameter and structural composition (12). In younger, healthy individuals, incident waves are reflected back toward the heart during diastole, which augments diastolic pressure (71). Reflected waves reaching the proximal aorta during diastole modulates an increase in myocardial perfusion, which aids the ability of oxygen rich blood to reach the heart muscle tissue. In people with stiff arteries, this pulse wave reflects backwards at a faster rate. This causes the reflected wave to reach the proximal aorta during the middle or end of systole, which augments systolic pressure and increases both pulse pressure and afterload (12, 24).

Pulse wave analysis (PWA) is a noninvasive method to analyze aortic waveforms. According to O'Rourke et al., analyzing aortic pulse waves requires a calibrated system via supine brachial blood pressure readings, an accurate recording of the radial pulse wave, and the computer-generated aortic wave form using a generalized transfer function (52). In this way, investigators can get an accurate estimate of the aortic waveform noninvasively, using the radial artery as the site of application for applanation tonometry.

One important descriptive value from PWA is the aortic augmentation index (AIx), which is defined as the augmentation pressure divided by pulse pressure (72). Typically, AIx is expressed as a percentage of pulse pressure. Augmentation pressure

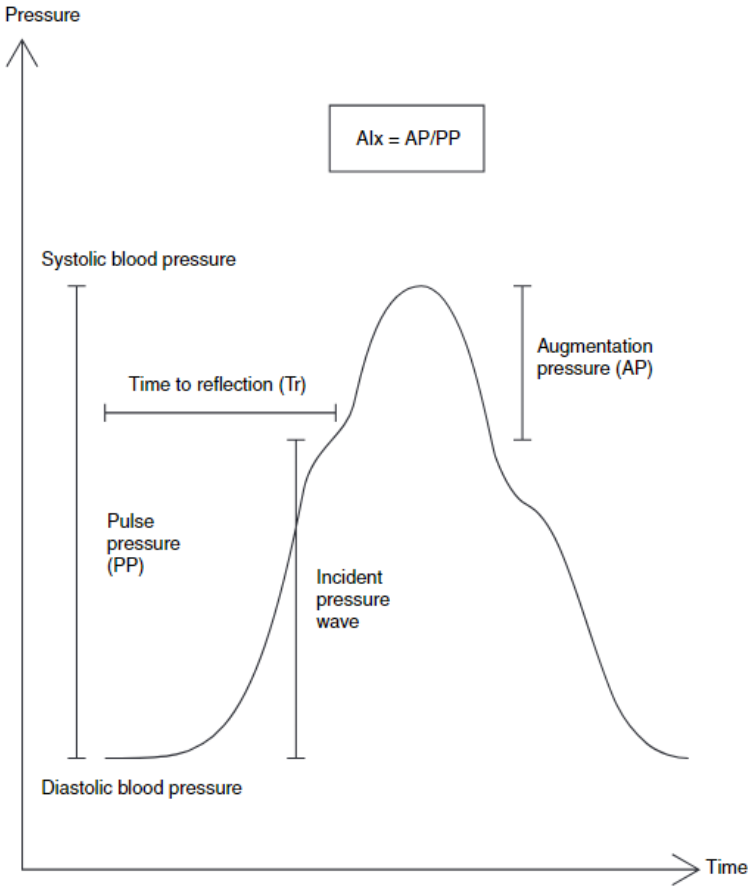

Figure 2. A visual depiction of the aortic pulse wave highlighting the relationship between the incident pressure wave, augmentation pressure (AP) and pulse pressure (PP) (29).

(AP) is the extra pressure added to the initial systolic inflection point (incident wave) from the reflected wave in the aorta, which can be influenced by when reflected waves arrive (i.e., systole vs. diastole) (10, 52). Pulse pressure is calculated simply as systolic blood pressure minus diastolic blood pressure and is influenced by stroke volume and aortic stiffness (72). Pulse pressure (PP) can be calculated from brachial blood pressure

readings, but values from the SphygmoCor system provide trusted estimates of aortic pulse pressure which has even greater clinical relevance (33, 46).

A final cardiovascular factor of importance relating to the pulse wave is aortic pulsatile load (aPL), defined as the product of heart rate and aortic pulse pressure (47). Pulsatile load describes the repetitive mechanical stress on arterial walls. Increases in PP, via an increase in SAP, influences the value of aPL. Over time, this increased stress can lead to elastin fatigue and smooth muscle hypertrophy and thus the development of stiffer arteries (32). Thus, increases in aPL can increase hemodynamic stress on end organs with delicate vasculature, such as the brain and kidneys.

1.2.2 Pulse Wave Velocity

Pulse wave velocity (PWV) is the gold standard method to assess and quantify arterial stiffness noninvasively as it is reproducible, simple, accurate, and robust (10). PWV is inversely related to the artery's elasticity; that is, a stiffer artery will propagate the pulse wave faster than a more elastic artery (10). Notably, it is an independent and powerful predictor of cardiovascular risk and all-cause mortality (10, 12, 70). Specifically, a 1 m/s increase in PWV is reported to be associated with a 10% greater risk in CV mortality and all-cause mortality (70). PWV is also a reliable predictor of future systolic blood pressure changes and development of hypertension in healthy individuals (10). PWV is faster in individuals with hypertension (43), suggesting that those who are hypertensive may have stiffer, more inelastic central arteries regardless of age (18). Mechanistically, stiffer arteries and a faster PWV reduce coronary perfusion due to augmented systolic pressure, which can lead to myocardial dysfunction (12). Similar to

PWA, central PWV is most commonly and noninvasively measured via applanation tonometry at the carotid artery and femoral artery sites, otherwise known as carotid-femoral PWV (cfPWV) (49).

1.3 Mindfulness

There is no one, current operational definition of mindfulness, although Jon Kabat-Zinn, a pioneer in current mindfulness practices and the founder of the mindfulness based stress reduction (MBSR) model, defines mindfulness as "the awareness that emerges through paying attention on purpose, in the present moment, and nonjudgmentally to the unfolding of experience moment by moment" (36). Mindfulness does not focus on changing the direct experience felt by an individual; rather, a key component of mindfulness is experiencing situations through non-judgmental awareness of the present moment and acceptance (11). Mindfulness practice can be considered a type of mental training with focus on reducing reactivity and similar states of mind that otherwise may perpetuate emotional distress (7).

The central practice of mindfulness is seated mindfulness meditation, in which individuals focus inward, usually on some aspect of the physical self (i.e., breathing) to stay focused and anchored in the present moment. Throughout the meditation practice, the mind will inevitably wander from the breath, but the individual simply notices this and brings the awareness back to the breath without judgment or rumination. This formal practice sets the foundation for informal, here-and-now awareness throughout daily living (7).

Recently, mindfulness has drawn considerable interest within the scientific community due to a variety of psychological and behavioral benefits. While mindfulness research is still rampantly gaining popularity, previous studies have shown promising results. A primary goal of mindfulness programs is mitigation of stress reactivity. One study found meditation practices may reduce stress reactivity in at-risk stressed populations, which in turn, may reverse or mitigate disease outcomes related to high levels of physiological stress and arousal (7).

Other studies have observed further benefits, including increases in attention post-meditation (17), cooperation and altruism (31), as well as decreases in binge and emotional eating (37). The wide scope of benefits mindfulness can offer strengthens the argument that mindfulness may be a clinically relevant and useful tool to improve the health and well-being of all individuals.

1.3.1. Mindfulness and Cardiovascular Health

Along with studied psychological benefits, mindfulness practices have proposed cardiovascular benefits. While mechanisms for the benefits of mindfulness on cardiovascular health are unknown, theoretical framework includes improved attentional control, improved emotional control, and self-awareness (42). These factors relate to the ability to enact and maintain beneficial lifestyle changes to improve health (i.e., quitting smoking).

A physiological mechanism to consider is the hypothalamic-pituitary-adrenal (HPA) axis. The implementation of mindfulness programs may be an effective way to decrease effects of overactivity from the HPA axis via decreasing the severity of the

stress response. In individuals with hypertension, 8-weeks of an MBSR course significantly reduced clinical seated resting systolic and diastolic blood pressure (29, 57). In another study, decreases in blood pressure were related to the improvements in anxiety and stress levels, although primarily for diastolic blood pressure (57).

Other potential mechanisms include slow breathing as a modulator for cardiovascular control (63). In formal mindfulness practices such as seated meditations and body scans, individuals are encouraged to deepen their breath and focus on how the breath feels as it is inhaled. Instructors may cue their participants to "send the breath" to specific areas of the body, such as the chest and belly, to provide more direct guidance on the sensation of the increased depth of breath. An assortment of previous studies have demonstrated a reduction in blood pressure, both in hypertensive (34) and healthy individuals (58) who undergo slow, controlled breathing practices. This breathing practice promotes the activation of the parasympathetic nervous system via the Vagus (X) nerve, resulting in vasodilation and therefore reductions in blood pressure (63).

It is important to note that MBSR programs are not intended to be substitutes for medication, but rather an adjunct to potentially mitigate high blood pressure and other detrimental cardiovascular variables (29). A combination of pharmacological and nonpharmacological treatments is typically required for optimal blood pressure control (63).

1.3.2 Acute Meditation

Most current mindfulness literature studies the effects of mindfulness meditation in the scope of longitudinal mindfulness training (i.e., MBSR). However, longitudinal 8-

week courses are not always plausible due to socioeconomic barriers such as the extended time commitment, potential monetary cost of the program, and transportation issues. To make mindfulness practices more inclusive to a wider population, acute bouts of meditation may be a solution without the commitment to a longitudinal program.

Brief mindfulness meditation practices have demonstrated similar psychological mind-body benefits. Previous studies have indicated that brief meditation improved self-reported psychological stress reactivity compared to a control group (75 minutes over 3 days) (16), increased executive attentional control in inexperienced meditators (10 minutes for 1 day) (51), decreases in the cortisol awakening response (360 minutes over 4 days) (4), and reduced pain and state anxiety (60 minutes over 3 days) (74). Additionally, an hour of guided mindfulness meditation reduced anxiety and aortic pulsatile load in a small cohort of individuals with mild to moderate anxiety (19). These results are noteworthy in that they suggest even small bouts of mindfulness meditation can positively influence an individual's health and wellbeing. While these psychological benefits are clear, there is limited literature discussing how a short practice of mindfulness meditation may influence physiological variables of the cardiovascular system such as arterial stiffness.

1.4 Decentering

Decentering is an aspect of mindfulness in which distant perspectives of thoughts and emotions are adopted. To analyze decentering, three proposed interrelated processes involving metacognition were developed (6). These processes include meta-awareness, or the awareness that thoughts or feelings are occurring; disidentification from internal

experience, or being able to separate oneself from internal states; and reduced reactivity to thought content, or demonstrating a diminished rumination of thoughts (5). In this way, decentering can be simply defined as understanding thoughts and emotions as transient, rather than permanent associations of oneself and accepting an objective perspective. For example, a distanced stance of emotions would be demonstrated by the statement, "I am aware I am feeling sadness," as opposed to "I am sad." This thought pattern dissociates negative feelings and emotions from the individual. Decentering, similar to other skills, can be improved with practice. Mindfulness practices, especially formal MBSR courses, can improve an individual's ability to decenter and improve overall life satisfaction (26, 28, 64).

The most commonly used method to measure the ability to decenter (trait decentering) is the Experiences Questionnaire (EQ) from David Fresco. The EQ is an 11-item questionnaire based on a Likert-type scale, with responses ranging from 1 (never or rarely true) to 5 (often or always true). This questionnaire measures the extent to which an individual can decenter in three ways: the ability to dissociate from thoughts, the ability to demonstrate self-compassion, and the ability to avoid habitual responses to negative stimuli (22). The EQ demonstrates good internal consistency with Cronbach's alpha of 0.83 (22). Additionally, the EQ performs similarly across a variety of factors, including sex, age, ethnicity/race, and previous meditation experience, suggesting that it provides an unbiased and comparable measurement of decentering among a diverse cohort (48).

1.4.1 Decentering and Anxiety

Enhancements in decentering via formal MBSR courses have been associated with a variety of health benefits, including an increased acceptance of pain (45), a decrease in depressive symptoms (38), and a reduction in anxiety symptoms (25-27). Improvements in all aspects of mindfulness typically occur, but anxiety tends to correlate specifically with decentering (25-27). While most of the causal mechanisms behind improved decentering remain unknown, a variety of studies have found an association between increases in decentering with lower anxiety scores.

Recently, decentering has been used as a tool in a variety of cognitive therapies for individuals suffering from anxiety disorders, such as generalized anxiety disorder (GAD), including emotion relation therapy and cognitive behavioral therapies. Hoge et al., concluded that individuals suffering from GAD who underwent a modified MBSR course showed a decrease in physiological anxiety symptoms, which was associated with an increase in decentering (26). Further research found similar results in GAD patients undergoing emotional regulation therapy without a formal MBSR course (53). These findings suggest the improvement of decentering may provide significant relief in anxiety and its associated symptoms.

1.4.2 Anxiety Inventories

Self-reported anxiety inventories are imperative for establishing objective anxiety values from individuals. These inventories are especially useful for evidence-based medicine and can aid physicians in the screening of mental disorders, including anxiety disorders, by assessing self-reported symptoms (61). To validate these screening tools,

Cronbach's alpha is used to determine the validity, comparability, and internal consistency of each psychometric inventory. Two of the most well-known and implemented inventories include the State Trait Anxiety Inventory (STAI) and the Beck Anxiety Inventory (BAI).

Charles Spielberger's STAI is an inventory that assesses both state anxiety and trait anxiety. State anxiety is defined as how the patient feels right now, in this moment. Trait anxiety relates to how an individual generally feels on a day-to-day basis. State and trait anxiety are polled on two separate forms. They are both 20-item questionnaires scored on a Likert-type scale, ranging from 1 (not at all) to 4 (very much so). When comparing psychometric inventories, the STAI's internal consistency reliabilities were stable, especially that of the state anxiety scale, suggesting a reliable objectification of anxiety (2). Cronbach's alpha for these questionnaires ranged from 0.86-0.95 (35).

The BAI is a 21-item questionnaire that explores how much a patient has been bothered by anxiety-related symptoms over the past week. The BAI is scored on a 4-point Likert-type scale from 0 (not at all) to 3 (severely). Individuals who scored below 36 had low to moderate anxiety, while a score of 36 or above suggested high levels of anxiety. The reliability for this psychometric inventory is excellent, determined by a high Cronbach's alpha between 0.90-0.94 (35).

1.5 Summary and Hypothesis

In summary, CVD and hypertension account for the greatest number of deaths worldwide. Stress and anxiety are associated with a physiological role in the stress

response, which can contribute to the prevalence of hypertension and arterial stiffness. Measuring arterial stiffness is a reliable, noninvasive way to accurately estimate the stiffness of the aorta and central arteries via PWA and PWV, which are independent predictors of cardiovascular mortality.

Mindfulness meditation, an ancient stress relief practice, may be a viable method to reduce stress and improve a variety of cardiovascular variables. Even short durations of mindfulness meditation are correlated with various psychosocial improvements. Decentering, an aspect of mindfulness, can be improved through mindfulness practices and is associated with decreased levels of stress and anxiety.

For Study 1, we hypothesized that a one-hour session of acute meditation would decrease state anxiety and cardiovascular variables such as aPL, PWV, and AIx when comparing pre-intervention to post-intervention data. For Study 2, we hypothesized that individuals inexperienced in mindfulness practices who have a greater ability to decenter would have a slower pulse wave velocity and thus more elastic arteries.

2. Study 1: Acute Meditation

2.1 Introduction

Cardiovascular diseases (CVDs) are chronic diseases relating to the heart and vasculature that will kill up to an estimated 23.6 million people per year by 2030 (21). One of the most important risk factors for cardiovascular disease is hypertension (55). In particular, younger, college-aged subjects with slightly elevated blood pressure are three times more likely to develop hypertension later in life (50). This increase in blood pressure above normotensive levels can significantly and negatively impact the heart and vasculature at a younger age. Specifically, the premature stiffening of large arteries can lead to incident hypertension, damage to the microvasculature of organs, and increase left ventricle strain (12, 47, 65). These effects are also amplified by increases in pulsatility (47).

Previous studies have concluded that amplified levels of psychological stress and anxiety are associated with increases in blood pressure (41, 59, 67). Therefore, the reduction of stress may be an appropriate measure to help decrease and control blood pressure, and thus improve cardiovascular health. Mindfulness practices, such as mindfulness meditation, have been highlighted recently for their studied improvements in psychological variables such as anxiety (9).

While previous longitudinal studies have been conducted related to mindfulness, there is less data on the impact of a shorter, acute meditation intervention on cardiovascular health. In fact, while a variety of mindfulness studies have been

conducted, very few have a focus on the potential physiological effects. Currently, it is not well known how a one-hour session of acute meditation may impact arterial stiffness. We hypothesized that reductions would occur between pre-intervention and post-intervention trials in regard to anxiety, pulse wave velocity, aortic pulsatile load, and aortic augmentation index.

2.2 Methods

All testing took place in the Clinical and Applied Human Physiology Laboratory in the Biological Sciences department at Michigan Technological University.

2.2.1 Subjects

Thirteen (13) individuals ranging from 18 to 28 years of age volunteered to participate in this study. Eight females (22 ± 1 years old) and five males (20 ± 1 years old) were enrolled. Volunteers were recruited primarily by email and flyer. To participate in this study, individuals had to not be pregnant, not be taking cardiovascular medication, have no history of diabetes, not smoke or vape, have no prior experience with mindfulness meditation, and have a BMI of less than 30 kg/m^2. All enrolled participants met the criteria for inclusion and were in good health. Participants read and completed a consent form and were informed they could discontinue the study at any time with no penalty. There was no monetary compensation for this study. Due to the COVID-19 pandemic, data collection was prematurely terminated. Two additional participants underwent the orientation session but were not able to return to the lab for the

intervention session due to university shutdown; therefore, those data could not be included for analysis.

2.2.2 Orientation session

During the orientation session, participants were familiarized with the laboratory setting, equipment, and informed about the study. Any questions or concerns about the study were answered by the primary investigator at this time.

Participants' height and weight were measured. A wall-mounted stadiometer was used to record height, while weight and body mass index were recorded with a digital scale (Tanita BC-418). Body mass index (BMI) was calculated as weight (kg) divided by height (m^2). Then, participants were instructed to sit in a chair and quietly relax with their legs uncrossed for five minutes to ensure accurate blood pressure measurements. Three seated and supine blood pressures were recorded and averaged with an automated sphygmomanometer (Omron HEM907-XL) to calibrate the SphygmoCor system. Heart rate (HR), pulse wave analysis (PWA), pulse wave velocity (PWV), aortic augmentation index (AIx), and aortic augmentation index normalized to 75 heart beats per minute (AIx@75), central pulse wave velocity, aortic pulse pressure (aPP), and aortic pulsatile load (aPL) were the cardiovascular measurements of interest for this study. All of the above variables except for aPL were recorded from the SphygmoCor system. Aortic pulsatile load was manually calculated as HR * aPP.

To begin cardiovascular data collection, participants were instructed to lie down in a relaxed supine position with the palm of their right hand supinated. Measurements were collected for PWA, AIx, and AIx@75 by placing the SphygmoCor tonometry probe

directly on the site of the radial artery and gently pressing down against dense connective tissue and the radial carpal bones. Recordings were taken for at least 10 full cardiac cycles while maintaining a stationary position with the probe. Acceptable recordings required an operator index of 75 or higher on the SphygmoCor software. Duplicate recordings were taken and averaged for analysis.

Central arterial stiffness measurements, otherwise known as carotid femoral pulse wave velocity (cfPWV), were recorded using the carotid artery and femoral artery sites. These sites were palpated and marked with a marker dot or piece of opaque tape for the carotid and femoral sites, respectively. The straight-line distance of each of these sites relative to the suprasternal notch were measured and recorded to the closest millimeter. Three ECG electrodes were placed on the skin, located under the right and left clavicles and on the left anterior and inferior portion of the ribcage to record heart rhythms and R-waves. These recorded R-waves were gated to carotid or femoral pressure waves to calculate the time it takes for a pulse wave to arrive at each specified site after the heart begins ejecting blood. PWV is determined simply by the change in distance (in meters) divided by the time (in seconds). These measurements were also duplicated and averaged for analysis.

To assess cfPWV with the SphygmoCor system, the straight-line distances and average supine blood pressure were entered into the software. Recordings were taken by placing the tonometry probe on the marked carotid site for at least 10 cardiac cycles, then again on the femoral site. The order was then reversed for another set of recordings. Data were deemed acceptable if the two recordings were within 1 m/s of each other and the

standard deviation of the PWV for each cardiac cycle between the two recording sites was less than 10.0%. These trials were then averaged for analysis.

Participants then completed the Spielberger State Anxiety Inventory (Form Y). The Spielberger State Anxiety Inventory is a 20-item self-reported measure of state anxiety, or how an individual feels right now, in this moment. This inventory uses a Likert-type scale, ranging from 1 (not at all) to 4 (very much so). Participants were then scheduled for the intervention session day which occurred approximately one week later.

2.2.3 Intervention session

Upon return to the laboratory for the second session, all blood pressure and cardiovascular measurements were repeated in the same way as the orientation session. Participants chose to either sit in a chair or lie down for the meditation intervention. Before beginning the mindfulness interventions, one overhead light was dimmed for relaxation purposes and participants were offered either a pillow for their head (supine) or a meditation cushion (seated).

First, a two-minute introduction to mindfulness meditation video (Meditation 101 - https://www.youtube.com/watch?v=rqoxYKtEWEc) was viewed. This video informs the participant about the benefits of mindfulness and how to practice mindfulness quickly and simply each day. Participants then listened to two guided meditations (Mindfulness Meditation in 20 Minutes - https://www.youtube.com/watch?v=64ZU2UCQdmQ; Body Scan, Palouse Mindfulness - https://palousemindfulness.com/meditations/bodyscan.html) for a total duration of 55 minutes. These body scans invited the participant to first focus on lengthening the breath. Then, the participants were cued to focus on individual

segments of their body and how they feel in that moment with constant attention on the breath.

Following the intervention, participants remained supine or sitting for a one-hour rest period. During this time, participants were allowed to read, draw, or listen to music, but were not permitted to use electronic devices with screens. At the 55th minute of rest, the participant repeated the Spielberger State Anxiety Inventory. Cardiovascular measurements were taken post-intervention after the 60th minute of rest.

2.2.4 Statistical Analysis

Statistical data analyses were completed by using IBM SPSS Version 20 (Armonk, NY). Normality was assessed prior to further analysis and assumptions were met. Data are expressed as mean ± standard error.

Repeated measures analysis of variance (ANOVA) were conducted to assess time effect changes in all cardiovascular variables at three time intervals: orientation, pre-intervention, and post-intervention. These variables include SAP, DAP, HR, PP, AIx, AIx@75, cfPWV, and aPL. Mauchly's Test of Sphericity was performed for each variable to determine whether sphericity could be assumed. Significance for within-subject effects for time were concluded assuming sphericity or using Greenhouse-Geisser. Additionally, post-hoc Bonferroni pairwise comparisons and paired t-tests were performed for any significant ANOVA data. Data were considered significantly different when $p<0.05$.

2.3 Results

Orientation, pre-intervention, and post-intervention variables (Table 1) were analyzed to ensure there were no differences in baselines between the two testing days. There were no significant differences between any orientation and pre-intervention measurements.

Table 1. Cardiovascular and anxiety variables comparing orientation, pre-intervention, and post-intervention measurements. Values are considered significant when $P < 0.05$.

Variable	Orientation	Pre-Intervention	Post-Intervention	P-value
SAP (mmHg)	111 ± 4	109 ± 4	109 ± 3	0.696
DAP (mmHg)	61 ± 2	62 ± 2	63 ± 2	0.527
HR (bpm)	62 ± 3	62 ± 3	58 ± 2	0.060
AIx	-4.6 ± 3	-3.6 ± 3	-4.5 ± 3	0.920
AIx @ 75	-8.4 ± 3	-11.5 ± 2	-13.0 ± 4	0.194
cfPWV (m/s)	4.8 ± 0.2	5.1 ± 0.2	4.9 ± 0.1	0.323
aPP (mmHg)	30 ± 2	29 ± 2	27 ± 1	0.157
aPL (mmHg)	1811 ± 138	1785 ± 127	1555 ± 113	0.037
State anxiety	35 ± 4	34 ± 4	32 ± 5	0.350

Values are reported as mean ± SE. Systolic arterial pressure (SAP), diastolic arterial pressure (DAP), heart rate (HR), aortic augmentation index (AIx), aortic augmentation index normalized to 75 heart beats per minute (AIx@75), carotid-femoral pulse wave velocity (cfPWV), aortic pulse pressure (aPP), and aortic pulsatile load (aPL).

There was a significant time effect for changes in aPL. Aortic pulsatile load remained relatively the same between orientation and pre-intervention sessions but decreased significantly after the meditation intervention as depicted in Figure 3. A post-hoc paired samples t-test was performed for aortic pulsatile load data due to its previous significance from the ANOVA test.

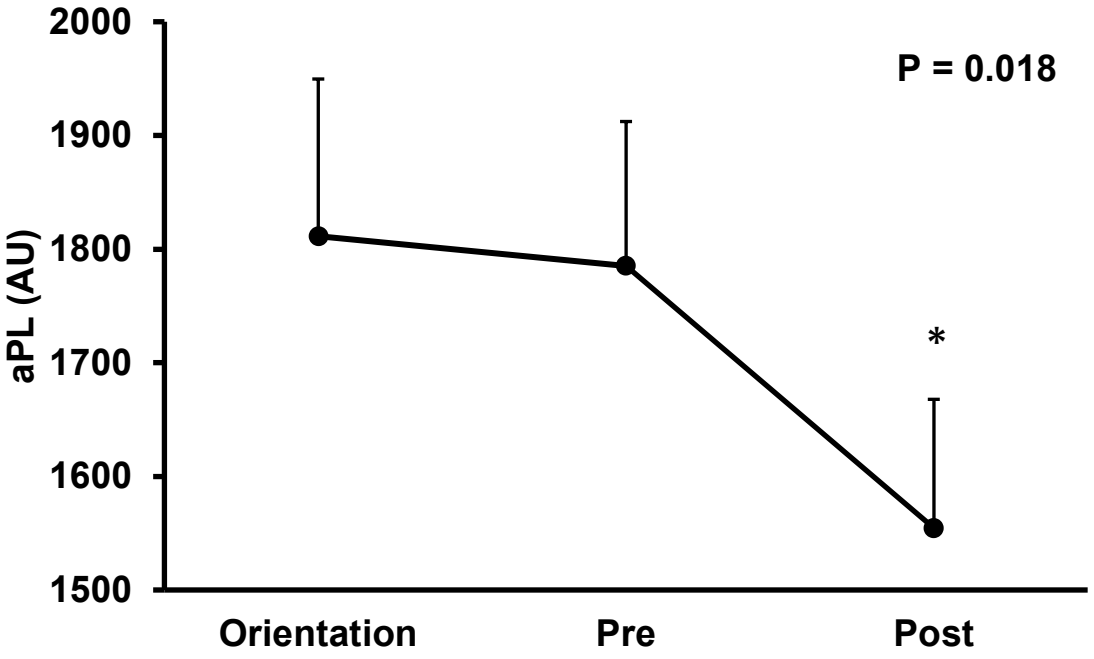

Figure 3. Time effect changes in aortic pulsatile load (aPL) at three time points. Data are significant when $P < 0.05$. *Difference from pre-intervention to post-intervention time points.

There were no significant differences between time points for other cardiovascular variables or for the state anxiety inventory. Aortic pulse pressure (Figure 5a) was unchanged between all time points. HR demonstrated a downward trend but was

not significant. SAP remained relatively constant, as well as DAP, AIx, AIx@75, and cfPWV. Last, there was no change in state anxiety from the state anxiety inventory.

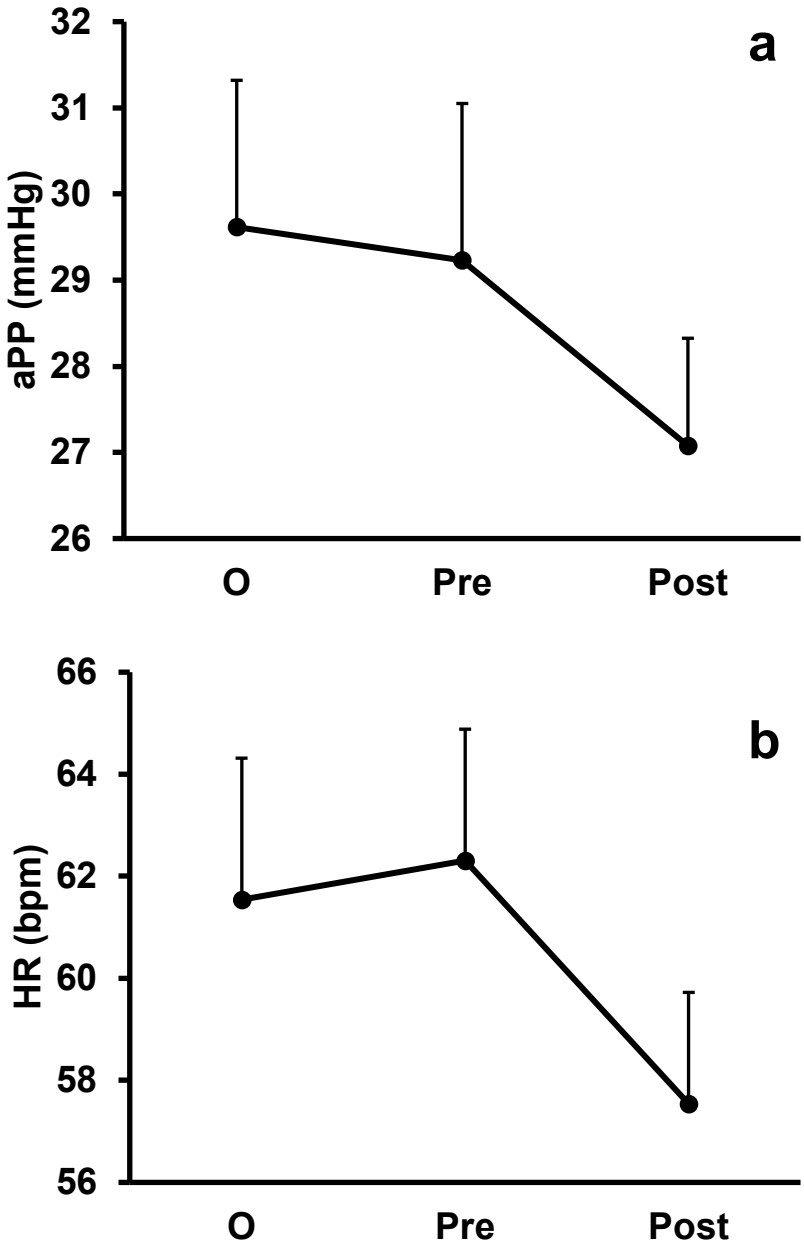

Figure 4a and 4b. Time-effect changes in (a) aortic pulse pressure (aPP) and (b) heart rate (HR) at three time points. Data are significant when $P < 0.05$.

2.4 Discussion

The major finding of this study indicates there is a significant decrease in aPL after a one-hour acute meditation intervention. This suggests that even short periods of mindfulness meditation can provide cardiovascular benefits to those who practice it. These results are consistent with a previous study in which mild to moderately anxious adults saw improvements in aPL after an hour long mindfulness meditation intervention (19).

Aortic pulsatile load is calculated by multiplying heart rate by pulse pressure. This value provides a good estimate of the mechanical stress placed on the proximal aorta, heart, and other organs such as the brain and kidneys. Increased pulsatile load can lead to cardiovascular dysfunction such as left ventricle remodeling and hypertrophy, which can lead to heart failure and death (3, 73). As pressure in the aorta increases, afterload increases, thus forcing the heart to have to eject blood with more force to overcome the pressure of blood within the aorta. The repeated, pulsatile mechanical stress of the blood against the proximal aorta can damage elastin fibers found in the arteries (32). As these fibers are damaged and replaced with stiffer, collagen fibers (10), their ability to expand and slowly recoil to accommodate the pulsatile flow of blood from the left ventricle diminishes.

Additionally, aPP, HR, AIx, AIx@75, and cfPWV were relatively unchanged. Because both aPP and HR decreased between pre-intervention and post-intervention, aPL was significantly reduced. Previous studies have demonstrated that pulse pressure is a better estimate than systolic blood pressure in regard to cardiovascular risk factors, as it

more closely represents the stress placed on the left ventricle and coronary arteries (60). Mechanistically, these reductions may also be partially due to a diminished stress response from the hypothalamic-pituitary-adrenal (HPA) axis due to a state of general relaxation.

Our study did not find any significant decreases in SAP or DAP before and after meditation. This is consistent with previous literature in which no blood pressure changes were detected in normotensive meditators after one hour of meditation (66). This could be due to the already normal or lower blood pressure levels in our participants. However, previous studies have found significant decreases in blood pressure after longitudinal 8-week mindfulness based stress reduction programs (29, 57) as well as transcendental meditation in hypertensive individuals (50). An acute meditation session may improve blood pressure better in hypertensive individuals but further research with this criterion should be completed.

While a variety of other acute studies have demonstrated significant decreases in anxiety or other negative emotions after meditation (16, 19, 50), we found that state anxiety remained unchanged from pre-intervention to 60 minutes post-intervention. We hypothesized there would be a larger, more significant difference in stress between pre-intervention and post-intervention. This discrepancy could be due to the waiting period between the end of meditation and the recording of cardiovascular variables 60 minutes later. Because our participants were inexperienced meditators, this relaxed state may not have carried over for the duration of the rest period.

Online guided mindfulness practices and courses can be used to further increase the accessibility of mindfulness meditation. Cavanagh, et al., concluded that an online-based, shortened program of two weeks found similar pre-test to post-test results, including significant increases in mindfulness and reductions in depression and anxiety symptoms, compared to a wait-listed control group (11). However, further studies should be completed comparing an online meditation cohort to an in-person cohort to determine the efficacy of this mindfulness medium. The potential availability of an online program can improve the availability of mindfulness meditation interventions platform to reach a wider variety of people.

In conclusion, short bouts of mindfulness meditation may be an effective way to decrease pulsatile load in the aorta. Acute meditation sessions may also decrease heart rate as well, but further investigation should be done with a larger sample size. Along with hypertension, CVDs have other attributable risk factors including arterial stiffness and pulsatility; thus, the addition of mindfulness meditation practices into daily routine may target other factors that antihypertensive drugs do not (55).

3. Study 2: Decentering and Arterial Stiffness

3.1 Introduction

The stiffness of large, central arteries in the body can be an important predictor of cardiovascular morbidity and mortality (69). Specifically, carotid-femoral pulse wave velocity (cfPWV) is a robust, accurate, and independent predictor of aortic stiffness and cardiovascular events (70). It can be assessed noninvasively with relative ease and is considered the gold standard method of measuring arterial stiffness (10). Overactivation of the stress pathway via psychological or psychosocial trauma can increase arterial stiffness prematurely (9). This can lead to adverse cardiovascular events at a younger age.

Mindfulness meditation is an ancient practice used to combat stress for centuries. While it has roots in Buddhist tradition, mindfulness meditation practices today are non-secular and inclusive. An important subcategory of mindfulness is decentering. Decentering is the ability to dissociate oneself from thoughts and emotions and accept a distant perspective (26). This mindset serves those practicing mindfulness as it combats rumination of negative thoughts by accepting them as transient, rather than permanent associations of oneself. The ability to decenter has been associated with an increased tolerance, or acceptance, of chronic pain as well as improvements in depressive symptoms and anxiety (22-24, 44).

While previous research on decentering has studied the psychological benefits, little is known about its relationship to cardiovascular physiology and specifically arterial stiffness. Therefore, the purpose of this study is to investigate the relationship between

the ability to decenter and arterial stiffness in individuals without previous meditation training. Previous studies have found that improvements in an individual's ability to decenter were associated with decreases in anxiety in individuals with generalized anxiety disorder (GAD) (26). Further, anxiety was robustly correlated with hypertension (29, 39, 40) and reductions in anxiety and stress were linked to positive cardiovascular outcomes such as decreases in blood pressure (56). Thus, we hypothesized that individuals who had a better ability to decenter would have lower arterial stiffness compared to those who had a lesser ability to decenter.

3.2 Methods

All testing took place in the Clinical and Applied Human Physiology Laboratory in the Biological Sciences department or in the Sleep Research Laboratory in the Kinesiology and Integrative Physiology department at Michigan Technological University. All protocols were approved by the Michigan Tech Institutional Review Board and all participants volunteered their written informed consent prior to any testing.

3.2.1 Subjects

A random sample of participants was recruited via flyers, email, and word of mouth. Fifty-five (55) healthy individuals enrolled in this study, with an average age of 24 ± 1 years old, and an average BMI of 24 ± 3 kg/m^2. One participant's data was omitted from final analyses due to the failure to complete the Experiences Questionnaire (EQ), thus data were collected for fifty four (54) participants.

36 males (24 ± 1 years old) and 18 females (23 ± 1 years old) completed the study. Participants were excluded from the study if they had previous experience with mindfulness meditation, defined as participating in a formal mindfulness meditation or mindfulness-based stress reduction (MBSR) course in the past. Additional exclusion criteria included smoking or vaping, taking cardiovascular or blood pressure medication, diabetes, or pregnancy. Participants volunteered to partake in this study and were informed they could withdraw at any point. There was no monetary compensation for completion of this study.

3.2.2 Experimental protocol

Participants were instructed to meet in the laboratory the day of testing having abstained from alcohol and caffeine consumption and exercise for at least twelve (12) hours prior to testing. Additionally, subjects were instructed to abstain from eating for at least three hours before measurements were recorded. Upon arrival, the investigator verbally explained the testing procedures and answered any questions volunteers had, and consent and exclusion criteria forms were completed. Participants also completed the 11-item Decentering Questionnaire (Experiences Questionnaire, EQ). This questionnaire measures an individual's current ability to decenter by gauging their ability to distance themselves from their thoughts, demonstrate self-compassion, and avoid habitual reactivity (i.e., rumination) to negative stimuli (22).

Subjects' heights were recorded using a wall-mounted stadiometer. Weight and body mass index (BMI) were recorded using an impedance analysis of body fat scale (Tanita BC-418). Then, participants were instructed to sit quietly and comfortably in a

chair for five minutes to achieve a relaxed, rested state. Three seated blood pressure recordings were measured and averaged using an automated sphygmomanometer (Omron HEM-907XL).

Triplicate supine recordings were measured and averaged following an additional five-minute rest period to calibrate the applanation tonometry system (SphygmoCor). Pulse wave analysis (PWA) at the radial artery site was performed in duplicate recordings to determine aortic augmentation index (AIx) and AIx normalized to 75 heart beats per minute (AIx @75). These values were averaged.

To measure heart rate and gate central and peripheral pulse waves, an electrocardiogram (ECG) was performed, attached to participants via three skin electrodes. cfPWV and peripheral (femoral artery to carotid artery) pulse wave velocity was performed to assess arterial stiffness at regional sites.

3.2.4 Statistical Analysis

Statistical data analyses were done using IBM SPSS Version 20 (Armonk, NY). Normality was assessed prior to further analysis. A median analysis was performed to compare arterial stiffness values in individuals with a high versus low ability to decenter. Independent samples t-tests were conducted to compare the two groups. Pearson correlations were performed to determine any associations between the independent variable (decentering) and dependent variables (AIx, AIx@75, cfPWV). Differences were considered significant if P-values were <0.05. Data are expressed as mean ± standard error.

3.3 Results

A median analysis was performed to categorize participants into either the "low decentering" (n=27) or "high decentering" (n=27) group. The high decentering group was composed of 10 females and 17 males, while the low decentering group contained 8 females and 19 males. The median score was 40, with the low decentering group scoring in the range of 26-39 on the Experiences Questionnaire (range of possible scores = 11 to 55) and the high decentering group scoring from 40-54.

Baseline anthropometrics and cardiovascular data are presented in Table 2 as the mean ± standard deviation. There were no significant differences in any anthropometric or cardiovascular data between participants in the low decentering group compared to the high decentering group, suggesting that any differences between the groups are not related to these variables.

Table 2. Participant anthropometrics and seated resting cardiovascular variables. Values are significant when $P < 0.05$.

Variable	Low decentering (n = 27; 24 ± 5 years)	High decentering (n=27; 23± 5 years)	P value
Height (cm)	172 ± 2	173 ± 2	0.731
Weight (kg)	74 ± 3	72 ± 3	0.559
BMI (kg/m2)	25 ± 1	24 ± 1	0.237
SAP (mmHg)	122 ± 2	119 ± 3	0.340
DAP (mmHg)	74 ± 2	69 ± 2	0.090
MAP (mmHg)	90 ± 2	86 ± 2	0.156
HR (bpm)	67 ± 2	71 ± 2	0.186

Values are mean ± SE. Body mass index (BMI), systolic arterial pressure (SAP), diastolic arterial pressure (DAP), mean arterial pressure (MAP), and heart rate (HR).

AIx and AIx@75 were similar in the high decentering group compared to the low decentering group, but this difference was not significant (Table 3). cfPWV was significantly lower in individuals who were better able to decenter than those who had a poor ability to decenter (5.6 ± 0.1 m/s vs. 5.1 ± 0.1 m/s, $P = 0.012$) as depicted in Figure 6.

Table 3. Median analysis of cardiovascular variables. Data are significant when $P < 0.05$.

Variable	Low decentering (n = 27)	High decentering (n = 27)	P value
AIx	4.7 ± 3	0.8 ± 2	0.319
AIx@75	-2.0 ± 3	-3.0 ± 3	0.780

Values are mean ± SE. Aortic augmentation index (AIx), aortic augmentation index normalized to 75 heart beats per minute (AIx@75), and carotid-femoral pulse wave velocity (cfPWV).

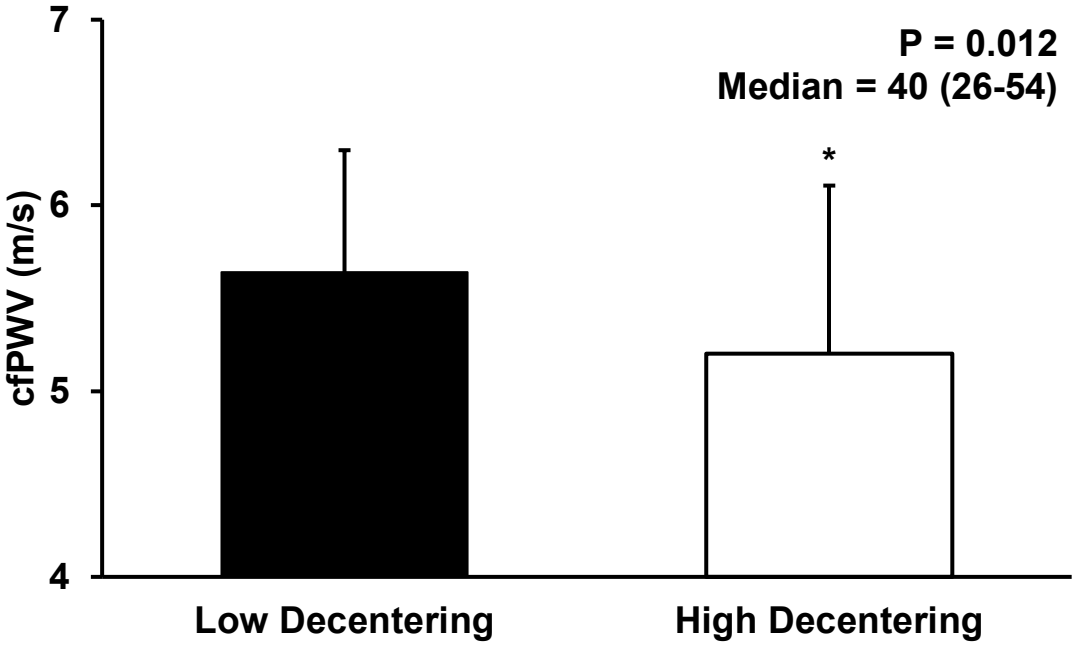

Figure 5. Carotid-femoral pulse wave velocity (cfPWV) as a function of a low versus high ability to decenter. Individuals in the high decentering group had a significant decrease in cfPWV compared to individuals in the low decentering group. *Data are significant when $P < 0.05$.

Decentering was also weakly correlated (r = -0.349, P = 0.01) with cfPWV as depicted in Figure 7. Both AIx and AIx@75 were not significantly correlated with decentering (AIx: r = -0.145, p=0.296; AIx@75: r = -0.066, P = 0.638).

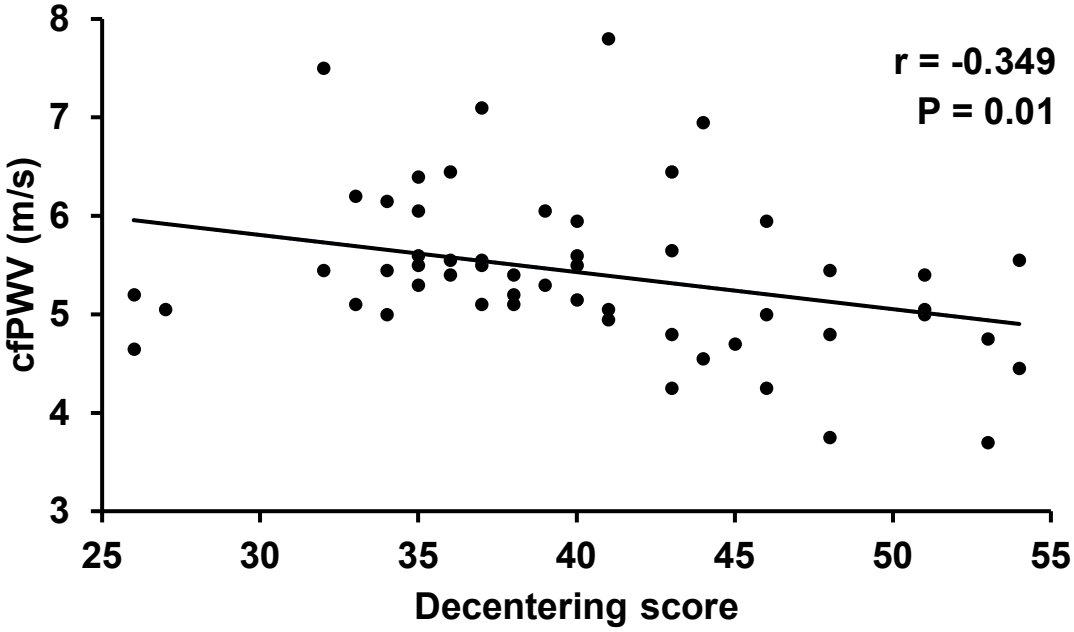

Figure 6. Correlational data for cfPWV and decentering. Higher decentering scores are correlated with slower central pulse wave velocity. Data are significant when $P < 0.05$.

3.4 Discussion

To our knowledge, this is the first study to report the relationship between an individual's ability to decenter and pulse wave velocity. Individuals who scored higher on the Experiences Questionnaire demonstrated a greater ability to decenter. This is inversely correlated with cfPWV, suggesting that a greater ability to decenter is significantly associated with a lower pulse wave velocity. Individuals in the high decentering group had a 0.5 m/s slower cfPWV than those in the low decentering group.

The significant reduction in cfPWV between the high decentering and low decentering groups suggests that developing this component of mindfulness may not only benefit people psychologically, but through cardiovascular health as well. The stiffening and lengthening of large central arteries are a normal process of aging (12). However, this process can be accelerated at a premature age due to a variety of factors, including hypertension, obesity, and excess stress (10). While some lifestyle changes, such as improving diet and increasing physical activity can help mitigate these, chronic stress can be more challenging to cope with. Individuals who are more chronically stressed may suffer from overstimulation of the sympathetic nervous system and the hypothalamic-pituitary-adrenal (HPA) axis. Among other functions, the HPA axis modulates the release of the stress hormone cortisol, which is essential for the stress response (8). However, issues arise when cortisol release becomes prolonged and chronic. This can cause inflammation, damage to the endothelium of the vessels, and smooth muscle hyperplasia, all of which contribute to increasing blood pressure and arterial stiffness (7, 22).

While this study measured an individual's unpracticed, inherent ability to decenter, decentering scores can be improved. Previous research has shown that an 8-week mindfulness-based stress reduction (MBSR) course improves people's ability to decenter (26). Decentering provides a positive coping mechanism for individuals who may be stressed or anxious. One study found that individuals who used task-oriented coping (focusing on the solution to a problem) were significantly less likely to have or develop hypertension than those who implemented emotional-oriented coping (ruminating on feelings) (19). This suggests that shifting coping mechanisms and

eliminating rumination on negative feelings and emotions are related to improvements in cardiovascular health. This is comparable to improving decentering.

In conclusion, the ability to decenter has important implications for cardiovascular health. Those who are better able to separate themselves from their thoughts and emotions tend to have more elastic arteries and a slower cfPWV. These results are relevant as previous studies with large sample sizes indicate that even a 1 m/s reduction in cfPWV may reduce the risk of cardiovascular events by 10% (70). This novel finding can offer new insight into the complicated relationship between the mind and body. It also strengthens the argument for the implementation of more MBSR-type courses into clinical settings in conjuncture with other lifestyle modifications.

4. Summary, Limitations, and Future Directions

4.1 Summary

Our results suggest that even an acute session of mindfulness meditation can have positive effects on the cardiovascular system. Additionally, individuals who are more mindful via decentering have slower pulse wave velocities. These novel findings advocate for the implementation of mindfulness interventions into daily life for improvements in cardiovascular health and wellbeing.

4.2 Limitations

While this research provides novel insights and considerations for mindfulness meditation and cardiovascular health, there are some limitations associated with the studies. First, due to the small number of participants of Study 1, there was no control group; however, the baseline data was comparable to the pre-mindfulness intervention, suggesting that any changes observed were in fact due to the meditation intervention. The small cohort also limits our ability to study possible sex differences in how they respond to a mindfulness intervention. The absence of a follow-up after the acute intervention is also a limitation. Having data on the longitudinal effects of a single session of mindfulness meditation would be insightful. Last, a lack of counterbalance in the order of measurements (i.e., providing the state anxiety questionnaire before versus after arterial stiffness measurements) may have provided a less accurate measure of state anxiety.

The use of seated, clinical blood pressure recordings may have impacted results. Ambulatory blood pressure monitoring is a more accurate representation of blood pressure on a day-to-day scale as it may fluctuate throughout a normal day due to everyday stressors as opposed to the controlled laboratory setting (42). To minimize this limitation, we took triplicate BP recordings after a standardized resting period at a similar time of day for between the two meetings.

Additionally, both studies enrolled primarily college-aged, healthy participants. This limits the scope of our studies in determining if mindfulness would be beneficial in older individuals who are more prone to arterial stiffness due to more advanced age.

4.3 Future Directions

Since physiology-focused mindfulness meditation research is still in its infancy, there are a variety of directions that this research can expand upon in the future. For acute meditation and continuing with previous research by Durocher, et al., a future consideration could be comparing the reliability of the BAI and STAI in individuals undergoing mindfulness interventions (19). Future studies should also include an in-person, instructor-led meditation cohort as a control to compare to an online recorded audio format. More invasive procedures could be implemented as well, including salivary and serum cortisol to gain insight on sympathetic and HPA axis response, or blood lipid levels. Additional routes with mindfulness research include studying the effects of acute mindfulness on sleep patterns. This could include cardiovascular variables throughout sleep such as blood pressure dipping.

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Appendix A: Raw Data for Study 1

A.1 Demographic Information

SUBJECT #	SEX	AGE (years)	HEIGHT (cm)	WEIGHT (kg)	BMI (kg/m^2)
1	F	19	160	65	25.2
2	M	18	173	52	17.5
3	F	28	152	35	15.2
4	M	23	185	90	26.2
5	F	23	162	65	24.7
6	M	19	173	63	21.1
7	F	19	178	70	22.1
8	M	21	173	72	24.1
9	F	22	163	59	22.2
10	F	19	163	47	17.7
11	M	19	180	93	28.7
12	F	20	173	62	20.7
13	F	26	170	63	21.8

A.2 Brachial Hemodynamics: Orientation

Subject #	SAP (mmHg)	DAP (mmHg)	MAP (mmHg)	HR (bpm)
1	97	50	66	59
2	100	60	73	76
3	94	55	68	43
4	117	63	81	50
5	132	74	93	58
6	128	60	83	77
7	116	59	78	69
8	128	64	85	71
9	99	58	72	59
10	98	50	66	56
11	124	72	89	72
12	123	68	86	68
13	84	58	67	63

A.3 Aortic Hemodynamics – Trial 1: Orientation

Subject #	SAP (mmHg)	DAP (mmHg)	MAP (mmHg)	PP (mmHg)	HR (bpm)
1	77	50	61	27	68
2	83	61	71	22	70
3	84	55	68	29	41
4	96	63	77	33	48
5	107	75	90	32	54
6	100	62	78	38	70
7	93	59	72	34	64
8	102	64	81	38	63
9	85	59	71	26	58
10	79	50	63	29	52
11	102	73	87	29	67
12	100	69	82	31	66
13	74	58	66	16	60

A.4 Aortic Hemodynamics – Trial 2: Orientation

Subject #	SAP (mmHg)	DAP (mmHg)	MAP (mmHg)	PP (mmHg)	HR (bpm)
1	77	50	62	27	75
2	83	61	71	22	75
3	83	55	67	28	41
4	97	64	78	33	49
5	107	75	91	32	58
6	100	62	77	38	74
7	94	59	73	35	69
8	102	65	83	37	65
9	85	59	72	26	54
10	79	50	64	29	54
11	102	73	86	29	72
12	100	69	83	31	67
13	75	59	67	16	60

A.5 Pulse Wave Analysis: Orientation

	Trial 1		Trial 2	
Subject #	AIx	AIx @ 75	AIx	AIx @ 75
1	-23	-26	-20	-21
2	-19	-21	-19	-18
3	21	5	19	3
4	-10	-23	-9	-22
5	-5	-15	-5	-13
6	-5	-8	-14	-15
7	-6	-11	-15	-18
8	-3	-8	-2	-7
9	12	2	11	1
10	-6	-17	1	-7
11	-7	-11	-11	-12
12	-10	-15	-9	-13
13	8	1	14	7

A.6 Pulse Wave Velocity: Orientation

	Trial 1		Trial 2	
Subject	Velocity (m/s)	SD	Velocity (m/s)	SD
1	4.3	0.3	4.2	0.3
2	5.7	0.4	6	0.5
3	4.9	0.5	4	0.2
4	4.7	0.2	4.2	0.3
5	5.5	0.3	5.7	0.3
6	4.4	0.4	5.1	0.4
7	4.4	0.2	3.9	0.2
8	4.9	0.3	5.1	0.4
9	5.3	0.2	5	0.2
10	3	0.2	3.4	0.2
11	6.5	0.6	5.3	0.5
12	6	0.4	5.8	0.6
13	4.4	0.3	4	0.2

A.7 Brachial Hemodynamics: Pre-Intervention

Subject #	SAP (mmHg)	DAP (mmHg)	MAP (mmHg)	HR (bpm)
1	97	51	66.3	62
2	102	62	75.3	69
3	93	54	67	35
4	117	63	81	50
5	111	72	85	58
6	115	63	80.3	74
7	119	61	80.3	68
8	129	75	93	67
9	99	56	70.3	71
10	131	69	89.7	72
11	120	63	82	67
12	89	62	71	71
13	111	62	78.3	66

A.8 Aortic Hemodynamics – Trial 1: Pre-Intervention

Subject #	SAP (mmHg)	DAP (mmHg)	MAP (mmHg)	PP (mmHg)	HR (bpm)
1	77	51	62	26	57
2	87	63	73	24	65
3	90	54	68	36	39
4	88	57	70	31	49
5	95	73	85	22	56
6	93	64	78	29	72
7	96	62	78	34	72
8	102	65	81	37	62
9	90	62	74	28	59
10	81	57	67	24	66
11	106	70	87	36	69
12	98	64	79	34	61
13	77	62	69	15	71

A.9 Aortic Hemodynamics – Trial 2: Pre-Intervention

Subject #	SAP (mmHg)	DAP (mmHg)	MAP (mmHg)	PP (mmHg)	HR (bpm)
1	77	51	63	26	60
2	87	63	74	24	67
3	90	55	70	35	39
4	88	57	71	31	53
5	96	73	85	23	58
6	94	63	76	31	67
7	96	62	76	34	65
8	103	65	84	38	69
9	90	62	75	28	65
10	82	57	69	25	67
11	105	70	86	35	72
12	97	64	79	33	61
13	77	62	70	15	72

A.10 Pulse Wave Analysis: Pre-Intervention

	Trial 1		Trial 2	
Subject #	**AIx**	**AIx @ 75**	**AIx**	**AIx @ 75**
1	-12	-19	-11	-20
2	-7	-10	-5	-10
3	34	-	34	-
4	-15	-26	-17	-30
5	4	-5	-1	-10
6	-9	-13	-6	-7
7	-3	-8	-2	-4
8	2	-1	-4	-10
9	-12	-17	-9	-17
10	-7	-10	-6	-11
11	-16	-18	-18	-21
12	1	-6	-4	-11
13	-5	-6	-2	-4

A.11 Pulse Wave Velocity: Pre-Intervention

	Trial 1		Trial 2	
Subject #	**Velocity (m/s)**	**SD**	**Velocity (m/s)**	**SD**
1	3.9	0.3	4.6	0.3
2	4.5	0.3	5	0.3
3	5.8	0.4	6.6	0.3
4	4.5	0.3	5.5	0.4
5	5.5	0.5	5.7	0.6
6	4.7	0.3	5.1	0.4
7	5.1	0.4	4.2	0.3
8	5.7	0.4	6	0.3
9	5.2	0.3	4.8	0.3
10	3.3	0.3	4.2	0.2
11	5.6	0.3	5.6	0.3
12	5.2	0.4	5.7	0.5
13	5.3	0.5	5.7	0.5

A.12 Brachial Hemodynamics: Post-Intervention

Subject #	SAP (mmHg)	DAP (mmHg)	MAP (mmHg)	HR (bpm)
1	99	54	69	63
2	95	58	70	53
3	93	54	67	46
4	111	61	78	44
5	115	80	92	49
6	104	57	73	59
7	118	57	77	71
8	117	62	80	56
9	111	64	80	62
10	96	57	70	59
11	122	65	84	65
12	119	71	87	80
13	111	73	86	64

A.13 Aortic Hemodynamics – Trial 1: Post-Intervention

Subject #	SAP (mmHg)	DAP (mmHg)	MAP (mmHg)	PP (mmHg)	HR (bpm)
1	80	54	65	26	60
2	79	58	68	21	53
3	83	55	68	28	51
4	90	61	73	29	44
5	101	80	90	21	48
6	84	57	68	27	58
7	93	58	75	35	70
8	94	63	76	31	56
9	91	65	77	26	62
10	79	58	67	21	56
11	99	66	83	33	67
12	94	65	78	29	55
13	100	77	88	23	63

A.14 Aortic Hemodynamics – Trial 2: Post-Intervention

Subject #	SAP (mmHg)	DAP (mmHg)	MAP (mmHg)	PP (mmHg)	HR (bpm)
1	79	54	63	25	65
2	79	58	67	21	53
3	84	55	67	29	41
4	90	61	72	29	43
5	101	81	91	20	50
6	84	57	70	27	60
7	92	57	73	35	72
8	94	63	76	31	56
9	91	65	77	26	61
10	79	57	67	22	61
11	99	66	82	33	63
12	94	65	78	29	58
13	100	77	88	23	65

A.15 Pulse Wave Analysis: Post-Intervention

	Trial 1		Trial 2	
Subject #	AIx	AIx @ 75	AIx	AIx @ 75
1	-19	-26	-19	-24
2	-15	-26	-12	-23
3	18	7	21	5
4	-17	-32	-24	-39
5	7	-6	7	-5
6	-10	-18	-5	-13
7	-4	-7	-8	-9
8	-6	-16	-6	-15
9	-13	-19	-7	-14
10	-10	-19	-11	-18
11	1	-3	2	-4
12	-11	-21	-9	-17
13	17	11	16	11

A.16 Pulse Wave Velocity: Post-Intervention

	Trial 1		Trial 2	
Subject #	Velocity (m/s)	SD	Velocity (m/s)	SD
1	5	0.4	5	0.5
2	4.2	0.2	4.8	0.1
3	4.7	0.3	5.2	0.1
4	4.4	0.3	4.9	0.5
5	5.3	0.3	4.9	0.2
6	4.9	0.3	4.6	0.5
7	4.1	0.2	4.8	0.3
8	5.3	0.4	5.7	0.5
9	5.3	0.3	6	0.8
10	4.3	0.2	4	0.2
11	5.7	0.4	5.3	0.2
12	5.1	0.3	4.8	0.2
13	5.2	0.3	4.6	0.3

A.17 Questionnaires: Orientation

Subject #	Resilience	State
1	157	20
2	159	27
3	-	54
4	168	20
5	145	41
6	144	24
7	107	34
8	122	59
9	167	49
10	150	29
11	133	46
12	166	25
13	156	26

A.18 Questionnaires: Pre-Intervention

Subject #	Decentering	State	Trait	Mindfulness
1	46	20	28	141
2	53	27	36	136
3	33	54	54	117
4	46	26	25	165
5	40	27	38	112
6	-	24	37	139
7	26	30	54	116
8	26	55	62	97
9	51	34	36	113
10	48	34	27	153
11	35	66	62	136
12	48	25	25	162
13	40	25	32	133

A.19 Questionnaires: Post-Intervention

Subject #	State
1	20
2	21
3	57
4	20
5	26
6	22
7	33
8	52
9	21
10	22
11	69
12	20
13	27

Appendix B: Summary Statistics for Study 1

B.1 Aortic Pulsatile Load

Repeated Measures ANOVA

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Greenhouse-Geisser
Time	0.670	4.401	2	0.111	0.752

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Partial Eta Squared	Sig.
Time	**Sphericity Assumed**	518786.369	2	259393.184	3.788	0.222	**0.037**
	Greenhouse-Geisser	518786.369	1.504	344928.598	3.788	0.222	0.053
	Huynh-Feldt	518786.369	1.672	310221.068	3.788	0.222	0.047
	Lower-bound	518786.369	1.000	518786.369	3.788	0.222	0.075

Pairwise Comparisons (Bonferroni)

(I) Time	(J) Time	Mean difference (I-J)	SE	Sig	95% Confidence Interval Lower Bound	95% Confidence Interval Upper Bound
1 (O)	2 (Pre)	26.115	88.833	0.774	-167.44	219.666
2 (Pre)	3 (Post)	230.558	84.52	0.018	46.404	414.711

Post Hoc Paired T-Tests

	Mean	Std. Deviation	95% Confidence Interval Lower	Upper	T	df	Sig. (2-tailed)
O aPL - Pre aPL	26.11538	320.29245	167.43541	219.66618	0.294	12	0.774
Pre aPL - Post aPL	230.55769	304.74206	46.40391	414.71147	2.728	12	0.018

B.2 Aortic Pulse Pressure

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Greenhouse-Geisser
Time	0.866	1.589	2	0.452	0.881

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Partial Eta Squared	Sig
Time	**Sphericity Assumed**	48.667	2	24.333	2.190	0.154	0.134
	Greenhouse-Geisser	48.667	1.763	27.606	2.190	0.154	0.141
	Huynh-Feldt	48.667	2.000	24.333	2.190	0.154	0.134
	Lower-bound	48.667	1.000	48.667	2.190	0.154	0.165

B.3 Heart Rate

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Greenhouse-Geisser
Time	0.886	1.336	2	0.513	0.897

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Partial Eta Squared	Sig
Time	**Sphericity Assumed**	170.462	2	85.231	3.172	0.166	0.060
	Greenhouse-Geisser	170.462	1.795	94.976	3.172	0.166	0.067
	Huynh-Feldt	170.462	2.000	85.231	3.172	0.166	0.060
	Lower-bound	170.462	1.000	170.462	3.172	0.166	0.100

B.4 Systolic Arterial Pressure

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Greenhouse-Geisser
Time	0.68	4.239	2	0.120	0.758

Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Partial Eta Squared	Sig
Time	**Sphericity Assumed**	36.974	2	18.487	0.368	0.030	0.696
	Greenhouse-Geisser	36.974	1.515	24.399	0.368	0.030	0.640
	Huynh-Feldt	36.974	1.688	21.902	0.368	0.030	0.662
	Lower-bound	36.974	1.000	36.974	0.368	0.030	0.556

B.5 Diastolic Arterial Pressure

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Greenhouse-Geisser
Time	0.875	1.463	2	0.481	0.889

Tests of Within Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Partial Eta Squared	Sig
Time	**Sphericity Assumed**	19.897	2	9.949	0.658	0.052	0.527
	Greenhouse-Geisser	19.897	1.779	11.188	0.658	0.052	0.511
	Huynh-Feldt	19.897	2.000	9.949	0.658	0.052	0.527
	Lower-bound	19.897	1.000	19.897	0.658	0.052	0.433

B.6 Aortic Augmentation Index

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Greenhouse-Geisser
Time	0.951	0.548	2	0.760	0.954

Tests of Within Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Partial Eta Squared	Sig
Time	**Sphericity Assumed**	7.782	2	3.891	0.084	0.007	0.920
	Greenhouse-Geisser	7.782	1.907	4.080	0.084	0.007	0.912
	Huynh-Feldt	7.782	2.000	3.891	0.084	0.007	0.920
	Lower-bound	7.782	1.000	7.782	0.084	0.007	0.777

B.7 Aortic Augmentation Index @ 75 BPM

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Greenhouse-Geisser
Time	0.998	0.024	2	0.988	0.988

Tests of Within Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Partial Eta Squared	Sig
Time	**Sphericity Assumed**	146.167	2	73.083	1.755	0.128	0.194
	Greenhouse-Geisser	146.167	1.996	73.245	1.755	0.128	0.195
	Huynh-Feldt	146.167	2.000	73.083	1.755	0.128	0.194
	Lower-bound	146.167	1.000	146.167	1.755	0.128	0.210

B.8 Carotid-Femoral Pulse Wave Velocity

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Greenhouse-Geisser
Time	0.863	1.619	2	0.445	0.880

Tests of Within Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Partial Eta Squared	Sig
Time	**Sphericity Assumed**	0.532	2	0.266	1.183	0.090	0.323
	Greenhouse-Geisser	0.532	1.759	0.303	1.183	0.090	0.320
	Huynh-Feldt	0.532	2.000	0.266	1.183	0.090	0.323
	Lower-bound	0.532	1.000	0.532	1.183	0.090	0.298

B.9 State Anxiety Inventory

Mauchly's Test of Sphericity

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Greenhouse-Geisser
Time	0.445	8.903	2	0.012	0.643

Tests of Within Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Partial Eta Squared	Sig
Time	**Sphericity Assumed**	86.000	2	43.000	1.096	0.084	0.350
	Greenhouse-Geisser	86.000	1.286	66.859	1.096	0.084	0.330
	Huynh-Feldt	86.000	1.374	62.587	1.096	0.084	0.333
	Lower-bound	86.000	1.000	86.000	1.096	0.084	0.316

Appendix C: Raw Data for Study 2

C.1 Demographics

SUBJECT #	SEX	AGE (years)	HEIGHT (cm)	WEIGHT (kg)	BMI (kg/m^2)
1	F	42	172	79.5	27.0
2	M	25	180	94.3	29.1
3	M	45	168	81.6	29.1
4	M	20	183	92.1	27.5
5	M	23	191	86.2	23.8
6	M	22	186	101.1	29.4
7	F	27	170	85.5	29.6
8	M	21	180	84.8	26.2
9	M	22	180	85.7	26.5
10	F	19	165	57.6	21.2
11	F	19	160	64.5	25.20
12	M	18	173	52.3	17.53
13	F	19	157	68.9	27.95
14	M	30	175	72.1	23.54
15	M	24	193	102.9	27.62
16	M	20	183	94.8	28.31
17	M	20	180	67.1	20.71
18	M	25	172	76.2	25.76
19	M	21	170	71.2	24.64
20	F	21	157	58.1	23.57
21	M	24	175	81.2	26.51
22	M	23	167	68.0	24.38
23	M	20	170	58.1	20.10
24	F	19	170	63.0	21.8
25	F	28	152	34.9	15.15
26	M	23	185	89.8	26.24
27	F	23	162	64.9	24.73
28	F	19	178	69.9	22.09
29	M	21	178	66.2	20.83

30	M	26	175	73.4	23.04
31	M	26	157	58	23.53
32	M	27	186	87	25.15
33	M	21	171	78	26.67
34	M	23	185	93	27.17
35	M	26	175	81	26.45
36	F	23	155	70	29.14
37	M	22	178	80	25.25
38	M	27	183	74	22.10
39	M	22	167	68	24.38
40	M	25	168	60	21.26
41	M	21	189	104	29.11
42	M	23	159	67	26.50
43	M	28	175	67	21.88
44	M	28	177	70	22.34
45	M	23	162	60	22.86
46	F	23	170	58	20.07
47	F	28	148	69	29.86
48	F	18	152	59	25.54
49	M	21	178	80	25.25
50	F	19	163	47	17.69
51	M	19	180	94.8	29.26
52	F	20	173	62	20.72
53	F	26	170	63	21.80
54	F	22	163	59	22.21

C.2 Brachial Hemodynamics

Subject #	SAP (mmHg)	DAP (mmHg)	MAP (mmHg)	HR (bpm)
1	122	81	95	61
2	125	66	86	67
3	108	63	78	42
4	115	56	76	73
5	137	69	92	66
6	129	63	85	57
7	111	67	82	62
8	130	63	85	59
9	123	63	83	79
10	115	71	86	74
11	97	51	66	62
12	102	62	75	69
13	116	82	93	76
14	114	64	81	64
15	112	68	83	80
16	140	80	100	91
17	126	66	86	49
18	121	56	78	63
19	113	57	76	64
20	119	74	89	70
21	121	70	87	80
22	115	56	76	66
23	131	76	94	76
24	109	59	76	72
25	93	54	67	35
26	117	63	81	50
27	111	72	85	58

28	119	61	80	68
29	120	60	80	61
30	121	64	83	71
31	111	64	80	57
32	124	64	84	57
33	118	58	78	58
34	130	69	89	66
35	121	65	84	66
36	107	64	78	63
37	127	56	80	65
38	122	54	77	74
39	110	60	77	64
40	112	65	81	76
41	131	61	84	50
42	139	71	94	76
43	108	66	80	57
44	107	59	75	57
45	115	66	82	78
46	141	79	100	67
47	107	68	81	58
48	120	66	84	73
49	112	53	73	65
50	99	56	70	71
51	131	69	90	72
52	120	63	82	67
53	89	62	71	71
54	111	62	78	66

C.3 Aortic Hemodynamics – Trial 1

Subject #	SAP (mmHg)	DAP (mmHg)	MAP (mmHg)	PP (mmHg)	HR (bpm)
1	118	82	98	36	63
2	101	67	83	34	55
3	99	63	79	36	44
4	92	57	73	35	70
5	108	69	86	39	62
6	108	63	82	45	62
7	113	81	97	32	72
8	104	64	82	40	57
9	98	64	78	34	75
10	97	72	86	25	74
11	77	51	62	26	57
12	87	63	73	24	65
13	106	83	94	23	76
14	95	64	79	31	62
15	99	70	85	29	72
16	115	82	96	33	88
17	102	66	82	36	51
18	104	56	77	48	63
19	90	57	72	33	63
20	107	76	92	31	68
21	100	72	88	28	83
22	90	57	72	33	67
23	110	77	96	33	73
24	92	61	77	31	69
25	90	54	68	36	39
26	88	57	70	31	49

27	95	73	85	22	56
28	96	62	78	34	72
29	99	60	78	39	64
30	99	65	83	34	70
31	98	64	80	34	52
32	107	65	84	42	57
33	93	58	72	35	58
34	105	69	84	36	60
35	98	66	82	32	67
36	94	70	83	24	60
37	98	56	74	42	60
38	97	55	74	42	72
39	89	61	73	28	63
40	99	66	83	33	77
41	102	61	78	41	52
42	110	72	89	38	73
43	99	67	81	32	57
44	93	60	76	33	59
45	94	66	79	28	76
46	126	81	103	45	64
47	98	68	83	30	56
48	97	67	81	30	68
49	87	53	66	34	61
50	81	57	67	24	66
51	106	70	87	36	69
52	98	64	79	34	61
53	77	62	69	15	71
54	90	62	74	28	59

C.4 Aortic Hemodynamics – Trial 2

Subject #	SAP (mmHg)	DAP (mmHg)	MAP (mmHg)	PP (mmHg)	HR (bpm)
1	118	82	98	36	64
2	101	66	82	35	63
3	98	63	78	35	45
4	94	58	76	36	74
5	108	69	83	39	59
6	108	63	84	45	60
7	113	80	97	33	62
8	105	64	83	41	58
9	98	64	79	34	78
10	97	72	85	25	75
11	77	51	63	26	60
12	87	63	74	24	67
13	106	83	94	23	74
14	95	64	79	31	63
15	99	69	84	30	77
16	115	82	96	33	88
17	101	66	82	35	50
18	103	57	78	46	67
19	89	58	72	31	64
20	110	76	93	34	71
21	100	71	85	29	75
22	91	57	73	34	70
23	109	77	93	32	71
24	90	60	76	30	70
25	90	55	70	35	39
26	88	57	71	31	53

27	96	73	85	23	58
28	96	62	76	34	65
29	98	61	78	37	70
30	100	65	83	35	72
31	101	65	82	36	50
32	108	64	83	44	58
33	93	57	70	36	56
34	104	70	83	34	66
35	98	65	80	33	66
36	93	70	82	23	67
37	98	57	76	41	62
38	99	54	72	45	60
39	89	61	73	28	79
40	102	67	85	35	75
41	103	61	77	42	54
42	111	72	90	39	72
43	99	66	80	33	56
44	93	60	76	33	58
45	94	67	81	27	86
46	125	81	103	44	72
47	95	68	81	27	60
48	97	67	82	30	68
49	87	53	66	34	63
50	82	57	69	25	67
51	105	70	86	35	72
52	97	64	79	33	61
53	77	62	70	15	72
54	90	62	75	28	65

C.5 Pulse Wave Analysis

	Trial 1		Trial 2	
Subject #	**AIx**	**AIx @ 75**	**AIx**	**AIx @ 75**
1	35	29	36	31
2	-5	-14	-7	-13
3	25	10	17	3
4	5	3	9	9
5	-12	-18	-14	-22
6	15	8	15	8
7	22	20	23	17
8	-10	-19	5	-4
9	-6	-6	-7	-6
10	-1	-1	-5	-6
11	-11	-20	-12	-19
12	-5	-10	-7	-10
13	18	18	17	17
14	3	-3	5	-1
15	11	12	14	13
16	-12	-6	-13	-6
17	-1	-13	-5	-17
18	18	12	18	14
19	3	-2	-2	-8
20	22	20	25	22
21	-1	3	-10	-10
22	-13	-17	-14	-17
23	5	4	-2	-4
24	10	7	2	0
25	34	-	34	-
26	-17	-30	-15	-26
27	-1	-10	4	-5
28	-2	-4	-3	-8
29	11	6	7	5
30	3	1	8	7
31	16	5	28	16
32	12	4	13	4
33	-14	-22	-16	-26
34	-16	-23	-16	-20
35	-3	-7	-8	-12
36	14	7	10	7
37	-1	-9	-4	-7
38	-4	-6	3	-4
39	-20	-26	-16	-19
40	19	19	26	26

41	-10	-21	-14	-24
42	-6	-6	1	2
43	21	13	21	12
44	16	8	15	7
45	-12	-12	-5	1
46	25	22	24	22
47	25	16	16	9
48	-9	-13	-6	-10
49	-13	-19	-12	-18
50	-6	-11	-7	-10
51	-18	-21	-16	-18
52	-4	-11	1	-6
53	-2	-4	-5	-6
54	-9	-17	-12	-17

C.6 Pulse Wave Velocity

	Trial 1		Trial 2	
Subject #	Velocity (m/s)	SD	Velocity (m/s)	SD
1	7.9	0.7	7.1	0.5
2	4.9	0.3	5.7	0.4
3	5.9	0.5	6	0.2
4	4.5	0.3	5.1	0.6
5	5.2	0.4	5.4	0.3
6	5.2	0.4	4.4	0.3
7	5.8	0.4	6.1	0.5
8	5.2	0.5	5	0.5
9	5.9	0.4	7	0.6
10	4.1	0.3	5	0.5
11	3.9	0.3	4.6	0.3
12	4.5	0.3	5	0.3
13	7	0.8	7.2	0.6
14	5.5	0.4	5.6	0.4
15	5.2	0.3	5.6	0.3
16	6.6	0.6	7.3	0.4
17	4.4	0.2	5.6	0.5
18	5.5	0.5	4.7	0.3
19	5.2	0.3	5.2	0.4
20	6	0.6	6.8	0.6
21	5.3	0.3	5.5	0.3
22	4.5	0.2	4	0.4
23	4.7	0.4	4.2	0.2
24	5.1	0.3	5.2	0.3
25	5.8	0.4	6.6	0.3
26	4.5	0.3	5.5	0.4

27	5.5	0.5	5.7	0.6
28	5.1	0.4	4.2	0.3
29	5.3	0.2	5.1	0.2
30	4.6	0.2	5.3	0.4
31	5.2	0.6	5	0.6
32	5.9	0.5	5.2	0.2
33	6.8	0.8	6.1	0.6
34	5.6	0.2	5.2	0.3
35	5.6	0.5	5.3	0.6
36	5	0.6	5.9	0.9
37	6.5	0.6	5.6	0.3
38	5	0.5	5.1	0.4
39	4.6	0.5	4.8	0.3
40	5.1	0.4	5	0.4
41	5.2	0.4	5.8	0.4
42	5.2	0.4	6.1	0.5
43	5.8	0.4	6.5	0.7
44	6.1	0.7	6	1.1
45	5.6	0.4	5.5	0.2
46	7.6	0.5	8	0.4
47	5.3	0.4	5.7	0.5
48	4.8	0.2	5.3	0.5
49	3.6	0.5	3.8	0.5
50	3.3	0.3	4.2	0.2
51	5.6	0.3	5.6	0.3
52	5.2	0.4	5.7	0.5
53	5.3	0.5	5.7	0.5
54	5.2	0.3	4.8	0.3

C.7 Decentering Questionnaire

Subject #	Decentering Score
1	32
2	39
3	46
4	48
5	35
6	43
7	40
8	38
9	43
10	44
11	46
12	53
13	37
14	54
15	51
16	44
17	34
18	37
19	26
20	35
21	38
22	43
23	54
24	40
25	33
26	46
27	40

28	26
29	38
30	41
31	33
32	37
33	36
34	36
35	32
36	34
37	35
38	41
39	45
40	51
41	35
42	43
43	34
44	39
45	36
46	41
47	37
48	27
49	53
50	48
51	35
52	48
53	40
54	51

Appendix D: Summary Statistics for Study 2

D.1 Median analysis

Independent samples t-test

		95% Confidence Interval				
	Mean	**Lower**	**Upper**	**t**	**df**	**Sig (2-tailed)**
Decentering	-11.22222	-13.50009	-8.94435	9.886	52	0.000
AIx	3.85185	-3.82591	11.52962	1.007	52	0.319
AIx @ 75	1.05556	-6.49252	8.60363	0.281	52	0.780
cfPWV	0.51432	0.11718	0.91146	2.600	51	0.012

D.2 Correlations

	Pearson correlation	**Sig. (2-tailed)**	**N**
Decentering	1		54
Aix	-0.145	0.296	54
Aix @ 75	-0.066	0.638	54
cfPWV	-0.349	0.010	53